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EXAMINER	
RAE, CHARLESWORTH E	
ART UNIT	PAPER NUMBER
1611	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/556,220	THOMPSON ET AL.	
	Examiner	Art Unit	
	CHARLESWORTH RAE	1611	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *07 November 2008*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-26, 35-37, 49-53* is/are pending in the application.

4a) Of the above claim(s) *9 and 21* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-8,10-20,22-26,35-37 and 49-53* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *10/11/07*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Applicant's response, received 11/07/08, to the restriction/election requirements, mailed 10/07/08, electing invention I (claims 1-26, 35-37, and 49-53) and glioblastoma cells as the cancer cell species, hydroxycitrate as the ATP citrate lyase inhibitor species, and phosphoenolpyruvate as the tricarboxylate inhibitor species, is acknowledged.

Applicant's preliminary claim amendment, received 11/07/08, is also acknowledged.

Status of the Claims

Claims 1-26, 35-37, 49-53 are currently pending in this application.

Claim 9 and 21 are withdrawn for being directed to non-elected subject matter.

Claims 1-8, 10-20, 22-26, 35-37, 49-53 are under examination.

Restriction/Election

Applicant's statement that all of the pending claims read on glioblastoma cells is acknowledged and made of record. Applicant's statement that claims 1-8, 10-20, 22-26, and 50-53 read on hydroxycitrate, while claims 1-3, 7, 16, 35-37, and 49-53 read on phosphoenolpyruvate is also acknowledged and made of record.

It is noted that since applicant did not distinctly and specifically point out any supposed errors in the restriction requirement, the election is treated as an election without traverse (MPEP § 818.03(a)).

The restriction requirements are made final.

REJECTIONS

Claim rejections – 35 USC 112 – Second Paragraph

The following is a quotation of the second paragraph of 35 USC 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 8 and 20 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite in that it fails to point out what is included or excluded by the claim language. This claim is an omnibus type claim.

Claims 8 and 20 recite the term "having a structure defined by one of the formulae or examples set forth in U.S. Patent No. 5,447,954," which renders the claimed subject indefinite when viewed in light of the specification because even though the specification discloses *"[t]here are many examples of ATP citrate lyase inhibitors in the art. Each of U.S. Patent No. 5,447,954 ..., which are each incorporated herein by reference, disclose compounds that are ATP citrate lyase inhibitors,"* it is not clear which particular formulae or examples disclosed in US Patent 5,447,954 is being specifically referred to by the term "wherein said ATP citrate lyase inhibitor is selected from the group consisting of compounds having a structure defined by one of the formulae or examples set forth in U.S. Patent No. 5,447,954" (specification, page 9, lines 8-21). Thus, an artisan skilled in the art would not be able to reasonable determine the metes and bounds of the claimed subject because it is not clear what the formulae and examples referred to in the above referenced term specifically mean.

REJECTIONS

Claim rejections – 35 USC 103(a)

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

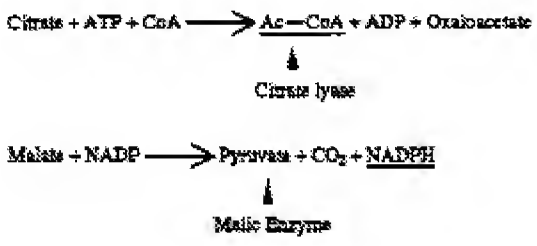
This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Claims 1, 4-8, 10-11, 14-15, 50-53 are rejected under 103(a) as being unpatentable over Kuhajda et al. (US Patent 5,759,837).

Applicant's election of glioblastoma as the cancer species is acknowledged. However, the instant claims do not recite the elected species and thus the scope is treated as recited (the genus).

It is noted that this rejection is being made under 103(a) because one would not immediately envisage specifically selecting hydroxycitrate from the various FAS inhibitors to treat cancer.

Kuhajda et al. (US Patent 5,759,837) teach methods of treating carcinomas that overexpress fatty acid synthase (FAS) or are dependent on endogenously synthesized fatty acid (e.g. ovary, prostate, breast, and colon) comprising administering a compound that inhibits FAS, including inhibitors for citrate lyase such as hydroxycitrate (abstract; col. 3, line 52 to col. 4, line 44; col. 7, line 29 to col. 8, line 31; col. 11, line 21 to col. 12, line 49, especially col. 11, line 60). Kuhajda et al. disclose that the fatty acid biosynthetic pathway in man is comprised of four enzymes: acetyl-CoA carboxylase, the rate limiting enzyme which synthesizes malonyl-CoA; malic enzyme, which produces NADPH; **citrate lyase**, which synthesizes acetyl-CoA; and fatty acid synthase, which catalyzes NADPH-dependent synthesis of fatty acids from acetyl-CoA and malonyl-CoA (col. 1, lines 45-51). Kuhajda et al. disclose the following enzymatic scheme showing that pyruvate and citrate lyase are involved in lipid synthesis (col. 16, lines 9-25):



Kuhajda et al. teach that since many tumor cells are extremely dependent on endogenous fatty acid synthesis, lower FAS activity levels need not exclude a specific tumor as a candidate for therapy with fatty acid synthase inhibitors (col. 7, lines 61-64). Kuhajda et al. teach that FAS expression and the growth inhibitory effect of inhibitors of the fatty acid synthetic pathway are not independent of the cell cycle and may be expected to be particularly effective in combination with chemotherapeutic agents that target rapidly cycling cells (col. 8, lines 53-65). Kuhajda et al. teach that a wide variety of compounds have bee shown to inhibit FAS and that selection of a suitable FAS inhibitor for the treatment of carcinoma patients is within the skill of the ordinary worker in this art (cols. 11-13, especially col. 11, lines 22-25 and lines 59-60). Kuhajda et al. teach that **any compound that inhibits fatty acid synthesis** may be used to inhibit tumor cell growth; a synergistic combination of at least one inhibitor of fatty acid synthesis and at least one inhibitor of either the enzymes which supply substrates to the fatty acid synthesis pathway or the enzymes that catalyze downstream processing and/or utilization of fatty acids may also be used (col. 10, lines 10-64; col. 16, lines 9-57). Kuhajda et al. teach that pharmaceutical compositions containing any of the inhibitors may be administered by parenteral, ..., topical, oral, ..., as necessitated by choice of drug, tumor type, and tumor location (col. 17, lines 32-37). Kuhajda et al. teach that the **dose and duration of therapy will depend on a variety of factors**, including the therapeutic index of the drug, tumor type, patient age, patient weight, and tolerance of toxicity and that generally the dose of the FAS inhibitor will be chosen to achieve serum concentrations from about 0.1 µg/ml to about 100 µg/ml; **the dose of a**

particular drug and duration of therapy for a particular patient can be determined by the skilled clinician using standard pharmacologic approaches in view of these factors (col. 17, lines 38-59). In addition, Kuhajda et al. teach that the presence of FAS in cells of the carcinoma may be detected by any suitable method, including activity assays or stains, immunoassays, ..., and the like (col. 7, line 65 to col. 8, line 22). Kuhajda et al. teach that most current methods of cancer therapy include treatment with chemotherapeutic agents that inhibit cell division or radiation therapy that disrupts DNA dividing cells that happen to be dividing or synthesizing DNA at the time of treatment and that there is a need for alternative treatment which more specifically target virulent tumor cells (col. 1, lines 36-43). Kuhajda et al. also teach that FAS inhibitors may also be used to **supplement a chemotherapeutic regime** (col. 8, lines 58-65).

Although Kuhajda et al. teach a method of treating cancer comprising administering the genus of FAS inhibitors (including ATP citrate lyase inhibitors), this reference does not teach the specific instantly claimed composition comprising an ATP citrate lyase or a tricarboxylate transporter inhibitor, or combinations comprising either subgenus.

It would have been obvious to a person of skill in the art at the time the invention was made to combine the teachings of the cited references to treat a patient with cancer, including cancers that are dependent on endogenously synthesized fatty acid and cancers that are independent of endogenously synthesized fatty acid, comprising administering any suitable FAS inhibitor (e.g. inhibitor of citrate lyase such as

hydroxycitrate), alone or in combination with another antineoplastic agent or radiation therapy, as taught by Kuhajda et al. to control the growth of the tumor cells. One would have been motivated to do so because Kuhajda et al. suggest that any suitable FAS inhibitor (e.g. hydroxycitrate) can be used to control the growth of cancer cells and that low FAS activity levels need not exclude a specific tumor as a candidate for therapy with fatty acid synthase inhibitors (col. 7, lines 61-64).

It is noted that the Markush claim language "the group consisting of: an ATP citrate lyase inhibitor, and a tricarboxylate inhibitor" as recited in claim 1 requires that the prior art teach either a composition comprising an ATP citrate lyase inhibitor or a composition comprising a tricarboxylate inhibitor, but not necessarily both, for purposes of this rejection. See MPEP 2173.05(h).

It is noted that the prior art teaches a method of treating patients with cancer and therefore the instantly claimed step of "identifying a patient with cancer" is implicitly taught by the prior art. Besides, it is routine in the oncology art to first diagnose a patient with cancer prior to administering anti-cancer treatment to said patient (claims 1, 10, and 52).

It is noted that Kuhajda et al. teach (-) hydroxycitrate, which reads on the term "an ATP citrate lyase inhibitor" as recited in claims 1, 4, 5, 6 and the term "a compound which inhibits the expression of ATP citrate lyase" as recited in claim 52. Claim 8 also recites (-) hydroxycitrate.

With respect to the term *"comprises cancer cells that have a high rate of aerobic glycolysis"* (claims 1, 11, and 53), it is the examiner's position that Kuhajda et al.

provides a general teaching of cancers, including cancers mediated by FAS, such that one would reasonably expect cancers mediated by FAS to be comprised of cancer cells that have a high rate of aerobic glycolysis since Kuhajda et al. teach that pyruvate is involved in lipid metabolism and pyruvate is also known to be involved in glycolysis (col. 16, lines 9-25).

With respect to the limitations regarding the therapeutic effective amount of an ATP citrate lyase (claims 1, 4, 5, 6, 8, 10, 52), Kuhajda et al. that the dose and duration of the FAS inhibitor therapy will depend on a variety of factors, including the therapeutic index of the drug, tumor type, patient age, patient weight, and tolerance of toxicity and that generally the dose of the FAS inhibitor will be chosen to achieve serum concentrations from about 0.1 µg/ml to about 100 µg/ml, which would reasonably be expected to be effective in inducing apoptosis in greater than 50% of cells in an in vitro apoptosis assay at a concentration of less than 1 mM since this concentration is also effective in inhibiting cancer growth, absent evidence to the contrary. Besides, it is the examiner's position that it would have been within the scope of skill and knowledge of an artisan skilled in the art at the time the invention was made to determine the effective amount of a FAS inhibitor (e.g. hydroxycitrate) to treat a patient with cancer, including the amount effective to induce apoptosis in greater than 50% of cells in an in vitro apoptosis assay at a concentration of less than 1 mM, as evidenced by the teaching of Kuhajda et al. the dose of a particular drug to treat a cancer patient can be determined by the skilled clinician using standard pharmacologic approaches (col. 17, lines 38-59).

With respect to the limitation regarding cancer cells that are not dependent on endogenously synthesized fatty acid (claims 7, 10), Kuhajda et al. teach that since many tumor cells are extremely dependent on endogenous fatty acid synthesis, lower FAS activity levels need not exclude a specific tumor as a candidate for therapy with fatty acid synthase inhibitors, and therefore it would have been obvious to a person of skill in the art at the time the invention was made to **empirically** treat a patient with cancer, including a patient with cancer cells not dependent on endogenously synthesized fatty acid, with a FAS inhibitor with a reasonable expectation of success absent evidence to the contrary (col. 7, lines 61-64).

With respect to claims 14 and 50, it would have been obvious to a person of skill in the art at the time the invention was made to treat a cancer patient with a FAS inhibitor and a different chemotherapeutic agent to control tumor growth. One would have been motivated to do so because the prior art suggest that FAS inhibitors may be used in combination with another chemotherapeutic agent, for example, to supplement the chemotherapeutic agent (col. 8, lines 58-65).

With respect to claims 15 and 51, it would have been obvious to a person of skill in the art at the time the invention was made to treat a cancer patient with a FAS inhibitor in combination with radiation therapy to control tumor growth. One would have been motivated to do so because the prior art suggest that FAS inhibitors may be use to supplement chemotherapeutic regimes (e.g. radiation therapy) (col. 8, lines 58-65).

Kudhajda et al. suggest that FAS inhibitors may be added to chemotherapeutic regimes (e.g. radiation therapy) to supplement said therapy (col. 1, lines 36-43; and col. 8, lines 58-65).

With respect to the preamble of claims 10 and 52, the prior art teaches a method of treating cancer and therefore implicitly teach a method of treating an individual identified as having cancer since diagnosis of a patient with cancer is the functional equivalent of identifying a patient with cancer.

Thus, it would have been obvious to a person of skill in the art at the time the invention was made to create the instant claimed invention with reasonable predictability.

Claims 2-3, and 12-13 are rejected under 103(a) as being unpatentable over Kuhajda et al. (US Patent 5,759,837), in view of Schroder et al. (Schroder et al. The role of 18F-fluoro-deoxyglucose positron emission tomography (18F-FDG PET) in diagnosis of ovarian cancer. Int. J. Gynecol Cancer. 1999;9:117-122).

The above discussion of Kuhajda et al. is incorporated by reference. Kuhajda teaches treating ovarian cancer. However, Kuhajda et al. do not teach PET imaging or 18—fluroro-deoxyglucose PET imaging (18F-FDG PET).

Schroder et al. (Schroder et al. The role of 18F-fluoro-deoxyglucose positron emission tomography (18F-FDG PET) in diagnosis of ovarian cancer. Int. J. Gynecol Cancer. 1999;9:117-122) teach that 18F-fluoro-deoxyglucose (or 18F-FDG) PET is a suitable method for detecting ovarian malignancies, particularly in patients with relapsed ovarian carcinoma (abstract). Schroder et al. state that the clinical significance and

usefulness of PET has been proven for a variety of other malignant tumors, .i.e. pancreatic carcinoma, glioma, etc. (page 117, col. 1, last two lines to col. 2, first five lines). Schroder et al. teach that 18F-FDG PET correctly metabolically differentiated primary ovarian tumors from inflammatory processes in 85.8 % of the patients studied (page 119, col. 2, last para., lines 1-10).

It would have been obvious to a person of skill in the art at the time the invention was made to combine the teachings of the cited references by using F-FDG PET imaging as taught by Schroder et al. to diagnose a patient with cancer (e.g. ovarian cancer). One would have been motivated to do so because Schroder et al. suggest that 18F-FDG PET imaging is useful in diagnosing malignant cancers by differentiating cancer cells from normal cells based on metabolic parameters and therefore one would reasonably expect that since the prior art teaches the same PET imaging using 18-fluorodexoyglucose to diagnose a patient with ovarian cancer that it would implicitly also determine the high rate of aerobic glycolysis in the cancer cells absent evidence to the contrary. Besides, since PET imaging is used to determine the metabolism of cancer cells and the rate of aerobic glycolysis is a measure of the metabolic process in cancer cells, one would reasonably expect that cancers diagnosed with PET imaging using 18-fluorodexoyglucose would be comprised of cancer cells with a high rate of aerobic glycolysis.

It is noted that both Schroder et al. and Kuhajda et al. teach cancers, including ovarian cancer, and Kuhajda et al. teach ovarian cancer as being a cancer that overexpress FAS.

Thus, it would have been obvious to a person of skill in the art at the time the invention was made to create the instant claimed invention with reasonable expectation of success.

Claims 16-20, 22, 25-26, 35-37, and 49 are rejected under 103(a) as being unpatentable over Kuhajda et al. (US Patent 5,759,837), in view of Bru et al. (US Patent 5,219,846).

The above discussion of Kuhajda et al. is incorporated by reference. However, Kuhajda et al. do not specifically teach the instant claimed combination of a ATP citrate lyase and a tricarboxylate transporter inhibitor.

Bru et al. (US Patent 5,219,846) teach methods for treating human tumors, particularly tumors that have become resistant to chemotherapy (e.g. colon, pancreas, stomach, bronchi, breasts), comprising administering an effective amount of phosphoenolpyruvic acid (abstract; col. 1, lines 41-64). Brue et al. teach that phosphoenolpyruvic acid is localized in the cells, and absent from the blood, under physiologic conditions (col. 1, lines 36-40).

It would have been obvious to a person of skill in the art at the time the invention was made to combine the teachings of the cited references by adding phosphoenolpyruvic acid as taught by Bru et al. to a citrate lyase inhibitor (e.g. hydroxycitrate) as taught by Kuhajda et al. for additive anti-tumor effects. One would have been motivated to do so because Kuhajda et al. suggest that FAS inhibitors (e.g.

ATP lyase inhibitors) can be combined with other chemotherapeutic agents and phosphoenolpyruvic acid as taught by Bru et al. is also a chemotherapeutic agent and both Bru et al. and Kuhajda et al. teach breast cancer (see In re Kerkhoven, 205 USPQ 1069 (CCPPA 1980).

With respect to the preamble (claim 16), the prior art teaches a method of treating cancer and therefore implicitly teach a method of treating an individual identified as having cancer since diagnosis of a patient with cancer is the functional equivalent of identifying a patient with cancer.

It is noted that the term phosphoenolpyruvic acid as taught by Bru et al. reads on the term "tricarboxylate transporter inhibitor" as recited in claims 16, 35, and 37. Claims 36 and 49 recite "phoshoenolpyruvate" which is the functional equivalent of phosphoenolpyruvic acid.

With respect to the term "therapeutically effective amount of a tricarboxylate inhibitor," Bru et al. teach phosphoenolpyruvic acid for use in an effective amount to treat cancers (e.g. breast cancer).

With respect to the limitations regarding the therapeutic effective amount of an ATP citrate lyase (claims 16, 17, 18, 19, 20, 22), the above discussion regarding the same discussion in connection with Kuhajda et al. is incorporated by reference.

With respect to the limitation regarding the *"high rate of aerobic glycolysis"* (claims 22, 37), the above discussion of this same limitation in connection with Kuhajda et al. is incorporated by reference.

With respect to the limitation regarding cancer cells that are not dependent on endogenously synthesized fatty acid (claims 19, 35), the above discussion regarding this same limitation in connection with Kuhajda et al. is incorporated by reference.

With respect to the limitation regarding a different anti-cancer compound (claim 25), the above discussion of this limitation in connection with Kuhajda et al. is incorporated by reference.

With respect to the limitation regarding radiation therapy (claim 26), the above discussion of this limitation in connection with Kuhajda et al. is incorporated by reference.

Thus, it would have been obvious to a person of skill in the art at the time the invention was made to create the instant claimed invention with reasonable predictability.

Claims 23-24 are rejected under 103(a) as being unpatentable over Kuhajda et al. (US Patent 5,759,837), in view of Bru et al. (US Patent 5,219,846), and further in view of Schroder et al. (Schroder et al. The role of 18F-fluoro-deoxyglucose positron emission tomography (18F-FDG PET) in diagnosis of ovarian cancer. Int. J. Gynecol Cancer. 1999;9:117-122).

It is noted that Kuhajda et al. and Bru et al. do not teach PET imaging or PET imaging using 18-fluorodeoxglucose.

The above discussion of Shroder et al. is incorporated by reference.

It would have been obvious to a person of skill in the art at the time the invention was made to combine the teachings of the cited references to using 18F-FDG PET imaging as taught by Schroder et al. to determine the cancers cells that have a high rate of aerobic glycolysis as taught by Kuhajda et al. One would have been motivated to do so because Schroder et al. teach PET imaging for determining the metabolism of primary ovarian tumors and Kuhajda et al. also teach ovarian cancer and therefore one would reasonably expect that since the prior art teaches the same PET imaging using 18-fluorodexoyglucose to diagnose a patient with ovarian cancer that it would implicitly also determine the high rate of aerobic glycolysis in the cancer cells since PET imaging is used to determine the metabolism of cancer cells and the rate of aerobic glycolysis is a measure of the metabolic process in cancer cells.

Thus, it would have been obvious to a person of skill in the art at the time the invention was made to create the instant claimed invention with reasonable expectation of success.

Conclusion

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Charlesworth Rae whose telephone number is 571-272-6029. The examiner can normally be reached between 9 a.m. to 5:30 p.m. Monday to Friday.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Sharmila G. Landau, can be reached at 571-272-0614. The fax phone

number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR.

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26 January 2009

/C. R./ Examiner, Art Unit 1611

/Sharmila Gollamudi Landau/

Supervisory Patent Examiner, Art Unit 1611